PALM INTRANET

Day : Friday
Date: 8/12/2005

Time: 14:10:19

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	**First Name**	
korneluk		Search

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Last Name	First Name	
holcik		Search

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Inventor Name Search

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Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
liston	p	Search

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Back to PALM | ASSIGNMENT | OASIS | Home page

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	4	"6348328"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:13
L2	509	korneluk.in. or holcik.in. or liston. in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L3	33	L2 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L4	9	apoptogen$.as.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L5	8	L4 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L6	225	xiap	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L7	144	L6 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L8	21855	inhibit SAME (transcription or translation)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L9	56	L7 and L8	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L10	877253	antisense molecules	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L11	12200	"antisense molecules"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L12	3866	L11 SAME L8	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59

L13	0	L12 and L2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L14	4	L12 and L6	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L15	20099	regulate WITH expression	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L16	2273	L15 and L12	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L17	9279	antisense WITH therapy	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L18	701	L16 and L17	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L19	32567	"sequence complementary"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L20	675	L19 and L18	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L21	675	L20 and "antisense molecule"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L22	15	"6107041".pn. or "6133437".pn. or "6537751".pn. or "6703491". pn. or "6783961".pn. "6673917". pn. or "6348328".pn. or "6300492".pn.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L23	2	"20020187946"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L24	2	"20040010136"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59

L25	2	"20040005584"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L26	2	"20020120121"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
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L28	509	korneluk.in. or holcik.in. or liston.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L29	225	xiap	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L30	21855	inhibit SAME (transcription or translation)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L31	877253	antisense molecules	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L32	12200	"antisense molecules"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L33	3866	L32 SAME L30	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L34	0	L33 and L28	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L35	20099	regulate WITH expression	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L36	9279	antisense WITH therapy	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59

L37	32567	"sequence complementary"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L38	2273	L35 and L33	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L39	701	L38 and L36	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L40	675	L37 and L39	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L41	675	L40 and "antisense molecule"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L42	33	L28 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L43	9	apoptogen$.as.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L44	8	L43 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L45	144	L29 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L46	56	L45 and L30	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L47	4	L33 and L29	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L48	144	L29 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59

L49	23	L27 and L28	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/12 13:59
L50	701	L38 and L36	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L51	88	L27 and L29	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/12 13:59
L52	19	L51 and IRES	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/12 13:59
L53	2273	L35 and L33	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L54	2	"6087173".pn.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/08/12 13:59
L55	509	korneluk.in. or holcik.in. or liston. in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L56	33	L55 and antisense	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L57	27	xiap and L56	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L58	3	L57 and "antisense therapy"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L59	0	L57 and "09/743,347"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L60	0	"09/743,347"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59

L61	27	xiap and L56	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59
L62	2	korneluk.in. and lacasse.in. and young.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/08/12 13:59

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FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 14:04:43 ON 12 AUG 2005
L1       112573 S HOLCIK?/AU OR KORNELUK?/AU OR LISTON?/AU OR YOUNG?/AU
L2         8602 S XIAP OR IAP OR (X-LINKED (S) APOPTOSIS)
L3        66939 S ANTISENSE
L4      1799763 S CANCER
L5           13 S L1 AND L2 AND L3 AND L4
L6            9 DUP REM L5 (4 DUPLICATES REMOVED)
L7          256 S L2 AND L3
L8           37 S L7 NOT PY>=2000
L9           15 DUP REM L8 (22 DUPLICATES REMOVED)
L10        1048 S ANTISENSE (2W) THERAPY
L11         250 S L10 AND (UNPREDICTABLE OR OBSTACLES OR DELIVERY OR TOXIC OR "
L12          67 S L11 AND REVIEW
L13          54 DUP REM L12 (13 DUPLICATES REMOVED)
L14          33 S L13 NOT PY<=2000
```

L6 ANSWER 1 OF 9 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
ACCESSION NUMBER: 2004287938 EMBASE
TITLE: Targeting endogenous inhibitors of apoptosis for treatment of **cancer**, stroke and multiple sclerosis.
AUTHOR: **Holcik M.**
CORPORATE SOURCE: Dr. M. Holcik, Apoptosis Research Center, Children's Hosp. of Eastern Ontario, University of Ottawa, 401 Smyth Road, Ottawa, Ont. K1H 8L1, Canada. martin@mgcheo.med.uottawa.ca
SOURCE: Expert Opinion on Therapeutic Targets, (2004) Vol. 8, No. 3, pp. 241-253.
Refs: 114
ISSN: 1472-8222 CODEN: EOTTAO
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
016 Cancer
022 Human Genetics
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20040722
Last Updated on STN: 20040722

AB The inhibitor of apoptosis (**IAP**) genes have emerged as probably the most important intrinsic regulators of apoptosis. The members of the **IAP** family are highly conserved in evolutionarily distant species and perform the critical role of binding to and inhibiting distinct caspases. This inhibition is mediated by discrete baculoviral **IAP** repeat domains that, in a domain-specific manner, inhibit either the initiator or executioner caspases. As such the function of IAPs lies at the very centre of virtually all apoptotic pathways. Since many, if not most, human pathologies involve aberrant apoptosis, the modulation of **IAP** levels or their activity offers huge therapeutic potential for treatment of various disorders. Indeed, available data suggest that the therapeutic downregulation of IAPs by **antisense** targeting or their adenovirally-mediated overexpression, can in fact be used to successfully modulate cell death. 2004 .COPYRGT. Ashley Publications Ltd.

L6 ANSWER 2 OF 9 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003325806 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12855663
TITLE: **Antisense** oligonucleotides targeting **XIAP** induce apoptosis and enhance chemotherapeutic activity against human lung **cancer** cells in vitro and in vivo.
AUTHOR: Hu YanPing; Cherton-Horvat Gabriele; Dragowska Visia; Baird Stephen; **Korneluk Robert G**; Durkin Jon P; Mayer Lawrence D; LaCasse Eric C
CORPORATE SOURCE: Department of Advanced Therapeutics, British Columbia Cancer Agency, Vancouver, British Columbia, Canada.
SOURCE: Clinical cancer research : an official journal of the American Association for Cancer Research, (2003 Jul) 9 (7) 2826-36.
Journal code: 9502500. ISSN: 1078-0432.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 20030713
Last Updated on STN: 20040421
Entered Medline: 20040420

AB Activation of programmed cell death in **cancer** cells offers novel and potentially useful approaches to improving patient responses to conventional chemotherapy. **X-linked** inhibitor of **apoptosis** (**XIAP**), is the most potent member of the

IAP gene family in terms of its ability to inhibit caspases and suppress **apoptosis**. In this study, we investigated the effect of **XIAP** down-regulation by **antisense** oligonucleotides (AS ODNs) on human non-small cell lung **cancer** (NIH-H460) growth in vitro and in vivo. In cultured H460 cells, G4 AS ODN was identified as the most potent compound. It down-regulated **XIAP** mRNA by 55% and protein levels up to 60% as determined by real-time quantitative reverse transcription-PCR and Western blotting, respectively, and induced 60% cell death. In contrast, the scrambled control ODN caused minimal **XIAP** loss and less than 10% cell death. Treatment with G4 AS ODN induced apoptosis as revealed by degradation of procaspase-3 and poly(ADP-ribose) polymerase proteins with significant nuclear DNA condensation and fragmentation. In addition, G4 AS ODNs sensitized H460 cells to the cytotoxic effects of doxorubicin, Taxol, vinorelbine, and etoposide. In animal models, administration of G4 AS ODN had significant sequence-specific inhibitory effects on H460 solid tumor establishment in a xenograft model. This antitumor activity was associated with an 85% down-regulation of **XIAP** protein in the tumors. In addition, the combination of 15 mg/kg G4 AS ODN with 5 mg/kg vinorelbine significantly delayed tumor establishment, more than either agent alone. These studies support the contention that **XIAP** is a viable target for **cancer** therapy in human non-small cell lung **cancer**.

L6 ANSWER 3 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:409132 BIOSIS
DOCUMENT NUMBER: PREV200200409132
TITLE: **Antisense** oligonucleotides targeting **XIAP** induce apoptosis and enhance therapeutic activity against human lung **cancer** cells when combined with anticancer drug in vitro and in vivo.
AUTHOR(S): Hu, Yanping [Reprint author]; Dragowska, Visia; **Korneluk, Robert**; Cherton-Horvat, Gabriele; Durkin, Jon; LaCasse, Eric; Mayer, Lawrence
CORPORATE SOURCE: Dept. of Advanced Therapeutics, British Columbia Cancer Agency, Vancouver, BC, Canada
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (March, 2002) Vol. 43, pp. 576. print.
Meeting Info.: 93rd Annual Meeting of the American Association for Cancer Research. San Francisco, California, USA. April 06-10, 2002.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Jul 2002
Last Updated on STN: 23 Sep 2002

L6 ANSWER 4 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:575599 BIOSIS
DOCUMENT NUMBER: PREV200100575599
TITLE: Modulation of IAPs for the diagnosis and **antisense** treatment of proliferative disease.
AUTHOR(S): **Korneluk, Robert G.** [Inventor, Reprint author]; Mackenzie, Alexander E. [Inventor]; **Liston, Peter** [Inventor]; Baird, Stephen [Inventor]; Tsang, Benjamin K. [Inventor]; Pratt, Christine [Inventor]
CORPORATE SOURCE: Ontario, Canada
ASSIGNEE: Aegera Therapeutics Inc., Verdum, Canada
PATENT INFORMATION: US 6300492 20011009
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct. 9, 2001) Vol. 1251, No. 2. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 2001
Last Updated on STN: 25 Feb 2002
AB Disclosed are diagnostic and prognostic kits for the detection and treatment of proliferative diseases such as ovarian **cancer**,

breast **cancer**, and lymphoma. Also disclosed are **cancer** therapeutics utilizing **IAP antisense** nucleic acids **IAP** fragments, and antibodies which specifically bind **IAP** polypeptides.

L6 ANSWER 5 OF 9 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001133428 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11145600
TITLE: Human ovarian **cancer** and cisplatin resistance: possible role of inhibitor of apoptosis proteins.
AUTHOR: Li J; Feng Q; Kim J M; Schneiderman D; **Liston P**; Li M; Vanderhyden B; Faught W; Fung M F; Senterman M; **Korneluk R G**; Tsang B K
CORPORATE SOURCE: Reproductive Biology Unit, Division of Gynecologic Oncology, Departments of Obstetrics and Gynecology, University of Ottawa.
SOURCE: Endocrinology, (2001 Jan) 142 (1) 370-80. Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010301

AB The inhibitor of apoptosis proteins (IAPs) constitutes a family of highly conserved apoptosis suppressor proteins that were originally identified in baculoviruses. Although **IAP** homologs have recently been demonstrated to suppress apoptosis in mammalian cells, their expression and role in human ovarian epithelial **cancer** and chemotherapy resistance are unknown. In the present study we used cisplatin-sensitive and -resistant human ovarian surface epithelial (hOSE) **cancer** cell lines and adenoviral **antisense** and sense complementary DNA expression to examine the role of **IAP** in the regulation of apoptosis in human ovarian **cancer** cells and chemoresistance. **Antisense** down-regulation of **X-linked** inhibitor of **apoptosis** protein (**Xiap**), but not human inhibitor of **apoptosis** protein-2 (Hiap-2), induced **apoptosis** in cisplatin-sensitive and, to a lesser extent, in -resistant cells. Cisplatin consistently decreased **Xiap** content and induced apoptosis in the cisplatin-sensitive, but not cisplatin-resistant, cells. Hiap-2 expression was either unaffected or inhibited to a lesser extent. The inhibition of **IAP** protein expression and induction of apoptosis by cisplatin was time and concentration dependent. Infection of cisplatin-sensitive cells with adenoviral sense **Xiap** complementary DNA resulted in overexpression of **Xiap** and markedly attenuated the ability of cisplatin to induce apoptosis. Immunohistochemical localization of the IAPs in hOSE tumors demonstrated the presence of **Xiap** and Hiap-2, with their levels being highest in proliferative, but not apoptotic, epithelial cells. These studies indicate that **Xiap** is an important element in the control of ovarian tumor growth and may be a point of regulation for cisplatin in the induction of apoptosis. These results suggest that the ability of cisplatin to down-regulate **Xiap** content may be an important determinant of chemosensitivity in hOSE **cancer**.

L6 ANSWER 6 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:421064 BIOSIS
DOCUMENT NUMBER: PREV200100421064
TITLE: **XIAP**: Apoptotic brake and promising therapeutic target.
AUTHOR(S): **Holcik, Martin**; Gibson, Hilary; **Korneluk, Robert G.** [Reprint author]
CORPORATE SOURCE: Solange Gauthier-Karsh Molecular Genetics Laboratory, Children's Hospital of Eastern Ontario, 401 Smyth Road, Room R306, Ottawa, ON, K1H 8L1, Canada

bob@mgcheo.med.uottawa.ca
SOURCE: Apoptosis, (August, 2001) Vol. 6, No. 4, pp. 253-261. print.
ISSN: 1360-8185.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Sep 2001
Last Updated on STN: 22 Feb 2002

AB The **X-linked** Inhibitor of **Apoptosis**, **XIAP**, is a key member of the newly discovered family of intrinsic inhibitors of **apoptosis** (**IAP**) proteins. IAPs block cell death both in vitro and in vivo by virtue of inhibition of distinct caspases. Although other proteins have been identified which inhibit upstream caspases, only the IAPs have been demonstrated to be endogenous repressors of the terminal caspase cascade. In turn, the caspase inhibiting activity of **XIAP** is negatively regulated by at least two **XIAP**-interacting proteins, XAF1 and Smac/DIABLO. In addition to the inhibition of caspases, recent discoveries from several laboratories suggest that **XIAP** is also involved in a number of other biologically significant cellular activities including modulation of receptor-mediated signal transduction and protein ubiquitination. **XIAP** is also translated by a rare cap-independent mechanism mediated by a specific sequence called IRES (for Internal Ribosome Entry Site) which is found in the **XIAP** 5' UTR. **XIAP** protein is thus synthesized under various conditions of cellular stress such as serum starvation and low dose gamma-irradiation induced apoptosis, conditions that lead to the inhibition of cellular protein synthesis. The multiple biological activities of **XIAP**, its unique translational and post-translational control and the centrality of the caspase cascade make the control of **XIAP** expression an exceptionally promising molecular target for modulating apoptosis. Therapeutic benefits can be derived from both the suppression of inappropriate cell death such as in neurodegenerative disorders and ischemic injury or in the activation of latent cell death pathways such as in autoimmune disease and **cancer** where apoptosis induction is the desired outcome.

L6 ANSWER 7 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:250005 BIOSIS
DOCUMENT NUMBER: PREV200100250005
TITLE: Modulation of IAPs for the treatment of proliferative diseases.
AUTHOR(S): **Korneluk, Robert G.** [Inventor, Reprint author]; MacKenzie, Alexander E. [Inventor]; **Liston, Peter** [Inventor]; Baird, Stephen [Inventor]; Tsang, Benjamin K. [Inventor]; Pratt, Christine [Inventor]
CORPORATE SOURCE: Ontario, Canada
ASSIGNEE: Apoptogen, Inc., Ottawa, Canada
PATENT INFORMATION: US 6133437 20001017
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Oct. 17, 2000) Vol. 1239, No. 3. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2001
Last Updated on STN: 19 Feb 2002

AB Disclosed are diagnostic and prognostic kits for the detection and treatment of proliferative diseases such as ovarian **cancer**, breast **cancer**, and lymphoma. Also disclosed are **cancer** therapeutics utilizing **IAP antisense** nucleic acids **IAP** fragments, and antibodies which specifically bind **IAP** polypeptides.

L6 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:202892 BIOSIS
DOCUMENT NUMBER: PREV200100202892
TITLE: Detection and modulation of IAPS for the diagnosis and treatment of proliferative disease.
AUTHOR(S): **Korneluk, Robert G.** [Inventor, Reprint author];

MacKenzie, Alexander E. [Inventor]; **Liston, Peter** [Inventor]; Baird, Stephen [Inventor]; Tsang, Benjamin K. [Inventor]; Pratt, Christine [Inventor]
CORPORATE SOURCE: Ontario, Canada
ASSIGNEE: Apoptogen, Inc., Ottawa, Canada
PATENT INFORMATION: US 6107041 20000822
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Aug. 22, 2000) Vol. 1237, No. 4. e-file. CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Apr 2001
Last Updated on STN: 18 Feb 2002

AB Disclosed are diagnostic and prognostic kits for the detection and treatment of proliferative diseases such as ovarian **cancer**, breast **cancer**, and lymphoma. Also disclosed are **cancer** therapeutics utilizing **IAP antisense** nucleic acids **IAP** fragments, and antibodies which specifically bind **IAP** polypeptides.

L6 ANSWER 9 OF 9 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 3
ACCESSION NUMBER: 2000360351 EMBASE
TITLE: Translational upregulation of **X-linked** inhibitor of **apoptosis** (**XIAP**) increases resistance to radiation induced cell death.
AUTHOR: **Holcik M.**; Yeh C.; **Korneluk R.G.**; Chow T.
CORPORATE SOURCE: R.G. Korneluk, Molecular Genetics, Research Institute, Children's Hosp. of Eastern Ontario, 401 Smyth Road, Ottawa, Ont. K1H 8L1, Canada
SOURCE: Oncogene, (24 Aug 2000) Vol. 19, No. 36, pp. 4174-4177. Refs: 20
ISSN: 0950-9232 CODEN: ONCNES
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 014 Radiology
016 Cancer
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20001026
Last Updated on STN: 20001026

AB Inhibitory regulators of apoptosis play a critical role in the responsiveness of turnout cells to cytotoxic agents. The **X-linked** inhibitor of **apoptosis** protein (**XIAP**) is a member of a novel family of Inhibitor of **Apoptosis** (**IAP**) proteins. Here we show that acute low dose ionizing irradiation results in the translational upregulation of **XIAP** that correlates with an increased resistance to radiation in non-small cell lung carcinoma. This upregulation is mediated by an internal ribosome binding mechanism via an IRES element located within a **XIAP** 5' UTR. Transient overexpression of **XIAP** rendered human carcinoma cells resistant to low dose γ-irradiation. By contrast, the **antisense** targeting of **XIAP** resulted in increased cell death following irradiation advocating a distinct role for **XIAP** in radiation resistant phenotype of human cancers.

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